duration of 3 1/2 years. About 15% of patients have a remission that lasts >5 years. The normoglycemic remission does not appear to be the result of weight loss.

Of the eight young Japanese men described by Nagasaka et al., seven appear to have a remission course similar to that noted in African-Americans by Banerji et al. (7).

The two major questions raised by both the studies in African-Americans and those in Japanese are as follows: *1*) What is the mechanism that is precipitating this syndrome? 2) Why is it prevalent in these particular races? A reasonable hypothesis can be formulated. African-American type 2 diabetic patients appear to have a greater β-cell insulin secretory deficiency than do Caucasian type 2 diabetic patients (8–10). The same is true for Japanese type 2 diabetic patients (11). When such populations become markedly insulin resistant, the stress on their insulin secretory reserve will be more severe. Any acute event then that either decreases insulin secretion or markedly increases insulin requirements would cause a severe imbalance between insulin requirements and insulin availability, thus precipitating diabetic ketoacidosis.

The factor causing insulin resistance in both African-American and Japanese populations appears to be central and/or generalized obesity. Factors that acutely inhibit insulin secretion could be neurally mediated factors or perhaps acute glucose toxicity (12) secondary to very large carbohydrate intakes that are rapidly absorbed. The acute event could be quite different from individual to individual. The high rate of normoglycemic remissions after intensive glycemic control observed in the Japanese and African-American subjects support the concept of an acute reversible inhibition of nutrient-mediated insulin secretion.

The extent to which this syndrome of diabetic ketoacidosis as the presenting event in type 2 diabetes is likely to increase is suggested by the marked increase in cases observed over the last several years in both populations. The potential role of high sugar consumption, while intriguing, needs further study.

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Comments on the Position Statements "Management of Dyslipidemia in Adults With Diabetes" and "Diabetic Retinopathy"

Position statements on the management of dyslipidemia in adults with diabetes (1) and diabetic retinopathy (2) were published in the January 1998 issue of *Diabetes Care*. I would like to make one practical point about each one. The position statement on dyslipidemia rightly recognizes that the risk from elevated LDL cholesterol concentrations outweighs the risk from elevated triglyceride (TG) levels. It follows then (as recommended) that the treatment for elevated LDL cholesterol levels (usually a statin) takes precedence over therapy for elevated TG levels. However, since many type 2 diabetic patients have TG levels >400 mg/dl, one cannot evaluate the LDL cholesterol concentration unless a direct measurement of LDL cholesterol is available. In this situation, (which is not uncommon in health maintenance organizations and other health care systems), the recommendation promulgated by the position statement is not tenable. One approach to this dilemma is to lower the elevated TG levels with gemfibrozil to <400 mg/dl and then evaluate LDL cholesterol concentrations. If it is above target levels, the fibrate is discontinued, and a statin is introduced. If TG levels rise again above 400 mg/dl, gemfibrozil is added back, and the patient is on combination therapy. Atorvastatin may be useful here instead of gemfibrozil, but I am unaware of published data regarding the response of elevated TG levels in diabetic patients.

My point regarding the diabetic retinopathy position statement concerns the recommendations for screening. Although seven-standard field stereoscopic 30° fundus photography is the gold standard for assessing diabetic retinopathy, very few patients currently have this modality available to them. The alternative of using a 45° nonmydriatic camera is dismissed unless a dilated eye examination is not available. However, there are a num-

ber of studies showing that nonmydriatic photography and dilated eye examinations are equivalent in screening diabetic retinopathy (i.e., clinical outcomes are very likely to be similar) (3–10). The problem of ungradable photographs in 10–20% of (mostly older) patients can be reduced considerably if the pupils are dilated on a subsequent attempt (8,10). As long as patients with any lesions noted on the photographs are referred to an ophthalmologist, serious problems requiring laser treatment will rarely be missed (9). Thus, less costly nonmydriatic photography is a very reasonable alternative for screening (a camera costs approximately $15,000; assuming a $100 cost for a complete dilated eye examination, the cost of the camera will be met after using it on 150 patients), especially for groups and organizations responsible for the diabetes care of large populations.

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Response to Davidson

Dr. Davidson (1) raises two interesting issues. The first relates to difficulties encountered in implementing guidelines for lipid management when LDL cholesterol cannot be estimated from a simple lipid screen. One option available is a direct measurement of LDL cholesterol. With new methods, this can be obtained for less than $150. As suggested by Dr. Davidson, treatment with a fibrate to lower triglycerides below 400 then reassessing LDL concentration provides a potential alternative in some practice settings. The LDL-based treatment goals suggested by the American Diabetes Association remain tenable, only the approaches to assay LDL cholesterol level differ. Several features of the former approach may make it preferable. First, therapy can be implemented in a more timely manner without requiring exposure of patients to a drug that may not be used for long-term lipid treatment. Second, and perhaps more importantly, it allows ongoing assessment of response to therapy in these patients in whom LDL lowering is found to be necessary. With ever-increasing evidence that long-term LDL lowering affects clinical outcomes in diabetic patients with increased LDL, ongoing measurement of LDL response assumes greater importance.

The second issue raised by Dr. Davidson is again important and practical. These recommendations were not written with a goal of dismissing the utility of the 45° nonmydriatic camera. Rather, they are written to recommend a standard of care. Responsibility (and liability) for both obtaining and interpreting the photographs obtained with the nonmydriatic camera would then reside with the physician (not an eye specialist) supervising the individual obtaining photographs. Some element of standardization in the reading of photographs would be desirable, and issues of conflict of interest regarding use of the diagnostic test for which the physician bills must be addressed. Unless these issues are resolved, it is difficult to endorse the nonmydriatic camera as a "standard of care." Used in the manner suggested by Dr. Davidson, i.e., referring all patients in whom any lesions indicative of retinopathy are noted, would appear to be safe and perhaps decrease patient inconvenience and overall medical cost.

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Hypoglycemia and Reduction of the Insulin Requirement as a Sign of Celiac Disease in Children With IDDM

It is well known that celiac disease is more frequent in patients with type 1 diabetes than in the general population, but the nonspecific nature of the presenting signs may cause a delay in diagnosis. Thus, Cronin and Shanahan (1) recommend that all IDDM patients should be screened for celiac disease not only at the diagnosis of diabetes, but every few years, since later tests may be positive.